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15.

THE SENSORY SYSTEM OF THE FACIAL NERVE AND ITS SYMPTOMATOLOGY[1]

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The subject which I have chosen for presentation this evening is the symptomatology of the sensory system of the seventh cranial nerve.

A preliminary statement of my views on this question was made before the American Neurological Association in 1907 (1); and certain special aspects of the same subject I have considered in previous communications, the "Herpetic Inflammations of the Geniculate Ganglion" (2) and "Otalgia Considered as an Affection of the Seventh Cranial Nerve" (3).

As an introductory statement I would emphasize the fact, that anatomically speaking the seventh nerve has long been recognized as a mixed nerve, having a sensory root and ganglion.

The investigations of Retzius (4), W. His (5), v. Lenhossek (6), Penzo (7), Sappolini (8), Van Gehuchten (9), and Dixon (10) have shown conclusively that the facial has a sensory ganglion the geniculate, a sensory root the pars intermedia of Wrisberg, as well as sensory fibers coursing in the chorda tympani, the great superficial petrosal nerve and the trunk proper of the seventh.

Modern authorities are agreed as to the nature and origin of the nerve of Wrisberg and the chorda tympani, opinions differ

[1] Presidential Address delivered before the New York Neurological Society, February 2, 1909.

however as to the nature, cause and derivation of the petrosal nerves. In my address this evening I shall outline the sensory development of the seventh nerve from a clinical standpoint and will attempt to give this system a definite and established group of sensory functions.

My attention was first directed to this subject by the study of certain forms of herpes zoster oticus, a distribution of herpes zoster which I referred to the geniculate ganglion of the facial nerve (Fig. 1). The herpetic pains associated with this condition

FIG. 1. Section through the intumescentia ganglioformis of the facial nerve showing the cells of the geniculate ganglion. (Weigert-Pal Method.)

were quite circumscribed and were definitely localized in the depths of the ear, the auditory canal and the mastoid region (the herpetic otalgia); and served to indicate the relation of the sensory facial to pain in the auditory mechanism (the otalgias). By this clinical method it was also possible to outline the zoster zone for the geniculate ganglion, which occupies an irregularly cone-shaped area on the auricle; the apex of which is represented by the tympanic membrane, the walls of the auditory canal, and the base by the concha, tragus, antitragus, anthelix and an adjacent marginal area.

Embryological methods had already shown the mixed nature of the facial nerve, its sensory system springing from the neural ridge, which also gives rise to the spinal ganglionic chain; furthermore anatomical studies in the lower vertebrate forms had shown the existence of a seventh nerve in which the sensory system was equal in importance to its motor, but which in the course of phylogenic development had undergone a very considerable shrinkage and diminution in importance, as it was replaced by the trigeminus. In man the sensory system is still concerned in the innervation of certain structures of the auditory mechanism, and a vestigial remnant still persists within the buccal cavity; and it is to the general sensory functions of this system which I shall ask your attention this evening.

The subject matter of my paper may be conveniently grouped under the following headings:

I. Historical résumé of sensory symptoms in facial palsy.
   (*a*) Pain in facial neuritis.
   (*b*) Anesthesia in facial palsy.
II. Anatomical considerations bearing on the sensory system of the seventh nerve.
III. The sensory symptoms and syndromes of the seventh nerve.
   (*a*) Herpetic inflammations of the geniculate ganglion and its complications.
   (*b*) Neuralgia of the seventh nerve (otalgia).
   (*c*) Pain and sensory disturbances in facial neuritis.
   (*d*) Facial spasms, twitchings and their etiological relation to the sensory system of the seventh.
IV. A scheme for the revision of the anatomical nomenclature of the seventh nerve.

Historical Résumé

As a proper introduction to this subject I shall recall some of the historical points of interest in connection with the symptomatology of the facial nerve, and more particularly those facts bearing on its sensory development.

To Sir Charles Bell, the eminent English anatomist and surgeon, is due the credit of first bringing into clinical prominence the facial nerve. Long before his time, however, the course of

the nerve and its anatomical relations were well known; and the origin in the medulla, circuitous path in the petrous bone and ramification on the face were described by the older anatomists.

Mangeti's (11) anatomy, published in the early part of the seventeenth century contains an admirable plate by Bartholomeus Eustachius showing the course of the nerve as we know it to-day, in which its ramifications on the face are clearly indicated and differentiated from those of the trigeminus. The differentiation of the terminal divisions of the fifth and seventh nerves at this period and up to the time of Bell, was not functional but merely an anatomical distinction; and it was not until the results of his epoch-making discoveries were reported to the Royal Philosophical Society (12) that the motor function of this nerve was clearly separated from the sensory function of the fifth.

It is not my purpose to discuss in detail these brilliant investigations, but it is of interest to note in passing that the earliest and most conclusive experiments made by Bell, and which later enabled him to separate the motor from the sensory roots of the cerebro-spinal system, were those carried out on the facial and trifacial nerves.

Bell's investigations, however, did not cease with his anatomical and experimental work, but were supplemented by very careful clinical studies of facial palsy, in which he elaborated this subject to an astonishing degree; recognizing most of the common etiological factors which we teach to-day, and also giving the points of differential diagnosis between facial palsies of central and those of peripheral origin.

In a description of one of his cases is the following statement, bearing on the involvement of the upper branch of the nerve in peripheral palsy (13).

"The patient could close the eyelid of the paralyzed side as well as the other, and when his nerve was stimulated by the hartshorn, or when he lifted the orbicularis oculi, the corrugator supercilii was in complete action, so that there was not here that heaviness of the upper part of the face so remarkable in paralytic cases. Here then is proof that those actions of the eyebrows which are found to be deficient when the portio dura is affected, are in the case of common palsy left entire."

In another case he writes: "Many instances will now occur to my readers of cases of paralysis of the face consequent upon a

local affection of the portio dura, which have been mistaken for an attack of apoplexy and the patient treated accordingly. In one case the patient having undergone the discipline of bleeding, purging and starving, and after having his head shaved and blistered, was suddenly cured by the bursting of an abscess in the ear."

Anatomically, Bell recognized the branch to the ossicles, the chorda tympani and its relations to the lingual branch of the fifth, the great superficial petrosal (Vidian nerve), the posterior auricular branch as the trunk emerges from the stylo-mastoid foramen and the peripheral distribution on the face. But as I have already indicated these bare anatomical facts had long been known to the older students of anatomy.

Since the time that facial palsy was shown to be dependent upon a lesion of the facial nerve, this structure has, for all practical purposes, been regarded clinically as a motor nerve. From time to time an occasional sensory symptom has attracted attention, but such sensory manifestations have been referred to one of the many neighboring sensory systems which converge and anastomose in this region.

In the present communication I shall not consider the mooted question of the central path of the fibers of the chorda tympani. The peripheral course and their origin in the cells of the geniculate ganglion have been definitely established, and the latest investigations also tend to show that the central path of these fibers is through the pars intermedia of Wrisberg. The facial has also important splanchnic functions, conveying vaso-motor and secretory impulses to the skin of the face, the lachrymal and salivary glands.

I will now pass to a consideration of various sensory symptoms which antedated my own investigations in this field.

Pain in Facial Palsy.—One of the first, if not the first, systematic study of a sensory symptom in facial paralysis was made by Dr. S. G. Webber (14), of Boston, in a paper entitled "Pain as a Symptom of Facial Paralysis and its Cause," published in the *Boston Medical and Surgical Journal* of December, 1876. In it is contained a careful description of the pain phenomena observed in six cases of facial palsy; the character of which was accurately described as well as its duration and area of distribution. The pain was localized in the ear and the mastoid region,

in some cases radiating to the face and occiput. Webber ascribed the pains to involvement of the peripheral filaments of the trigeminus nerve, and of the auricular branch of the vagus as it crosses the trunk of the facial in the lower portion of the Fallopian canal. He also suggested the possibility of recurrent sensory fibers of trigeminal origin passing backwards to the facial through the Vidian or greater superficial petrosal nerve.

Ten years later pain in facial palsy was made the subject of a thesis by Testaz, entitled "Paralysie douloureuse de la septieme paire" (15). Testaz collected reports of fifteen cases, in all but two of which pain was present. The pain as in Webber's original description was localized chiefly in the region of the ear and mastoid, which sometimes radiated to the face and occiput. He likened it in character to the pain of sciatica, and from its duration and severity, attempted to formulate a method of prognosis as to the severity of the paralysis. A *benign* form was recognized in which the pain preceded the palsy by a few hours up to one day, and a *severe* form in which several days or a week of pain elapsed before the appearance of the palsy.

Testaz, like Webber, referred the pain to neighboring sensory nerves. In all subsequent monographs treating of facial palsy the frequent occurrence of pain has found mention and its presence has been explained by a coincident involvement of adjacent sensory systems.

Anesthesia in Facial Palsy.—Besides the subjective symptom—pain—various types of objective sensory disturbances have been observed in the course of facial palsy.

In 1891, Frankl-Hochwart (16) directed attention to a peculiar form of sensory disturbance in facial paralysis. This consisted in a diminished sensibility of the entire face on the paralyzed side, in some cases including the tongue and mucous membrane. Sensation was not lost in this group of cases, but was merely diminished or obtunded in the area involved; it was of very short duration, and was present in eight of the twenty cases reported. He suggested that a coincident involvement of the terminals of the trigeminus nerve might be the underlying cause, but thought it also probable that the seventh nerve conveyed sensory fibers to the face. Here then was an attempt to attach a definite sensory function to the seventh nerve, but the very nature of the sensory distribution renders such a theory

hardly tenable; for it would be difficult to conceive of sensory fibers passing to the face in such numbers and so evenly distributed as to cause a uniform hypesthesia of the entire paralyzed area including the mucous membrane and the tongue, and even less so, as the phenomenon is not frequent.

Donath (17) in a recent contribution to this subject, based on a series of forty-three cases, found the hypesthesia of the face as described by Frankl-Hochwart in twenty cases. In fifteen of these, however, the diminished sensibility was not confined to the face alone but included the whole half of the body as well; it was therefore a hemi-hypesthesia, and as such could not be referred to the facial nerve. He recalls the normal physiological difference in the acuity of perception on the two sides of the body, and suggests this as a possible explanation.

It is also well known that hysterical hemi-hypesthesia in suggestible subjects is by no means rare. In the five remaining cases in which the sensory disturbance was confined to the face he regards it as a trigeminal manifestation, and probably due to coincident involvement of its terminals.

In order to show the variability and relative frequency of this symptom, Koster's (18) series of forty-one cases may be mentioned in which facial hypesthesia was absent; also Schieber's (19) series of fifty-six cases in which it was present in twenty-eight. Remak and Flatau (20) in a series of two hundred cases state that no sensory disturbances were found excepting in those cases complicated by herpes zoster, or hysteria.

Gowers (21) disposes of this type of hypesthesias of the face in the following manner: "Sensation in the face is generally unaffected, but I have several times in early and severe cases noted a very slight diminution in the sensitiveness of the skin especially on the cheek; the cause may be an alteration in the function of the nucleus of the fifth nerve due to the diminution of the impressions that are normally produced by muscular action. This is more probable than that the facial nerve sometimes conveys sensory fibers to the skin."

I may add that in thirty cases of facial palsy which I have examined personally for disturbances of sensation, this form of hypesthesia was present in only two. In these two cases the slightest touch upon the affected side was promptly perceived and it was only by careful comparison with the non-paralyzed

side that any difference became apparent in the acuity of sensibility. This was also true of the pain and temperature sense. And in my opinion a disturbance of this character, not hysterical, finds a more logical explanation in the functional theories of Gowers and Donath.

Another form of sensory disturbance is that described by Gowers (21), which consists of an area of hypesthesia in the concha of the ear. Gowers's description reads as follows:

"I have occasionally found in early cases an area of anesthesia on the front and back of the concha in the region of skin supplied by a nerve given off by the facial as it emerges, and which is probably derived from the fifth nerve." Here again, true to the tradition that the seventh nerve is a motor nerve, an adjacent sensory nerve, the trigeminus, is sought to explain the area of anesthesia. This particular area, it will be observed, lies within the zoster zone as outlined in my studies of herpes zoster oticus and represents the sensory distribution of the seventh nerve in the ear.

Hypesthesia in the Trigeminal Area of the Tongue in Facial Paralysis.—Another form of objective sensory disturbance was noted as early as 1876 by Bernhardt (22), which consisted of a hypesthesia of the tongue in the trigeminal area. This symptom is of rare occurrence but has been confirmed from time to time. In Schieber's series of fifty-eight cases it was observed but once. Koster found it absent in twenty-seven cases. In one hundred and thirty cases reported from Mendel's clinic (23) it was mentioned in only three. The rarity of this symptom may be more apparent than real as the disturbance of sensibility is very slight and requires care to demonstrate its presence. Furthermore, subjective and objective disturbance of the taste sensation may hamper its proper demonstration.

In connection with this form of sensory disturbance, Harvey Cushing (24) has made an interesting observation in his elaborate and most conclusive study of the trigeminal field after Gasserian ganglion extirpation. Cushing found that while there is anesthesia to touch, pain and temperature on the anterior two-thirds of the tongue after ganglion extirpation, a crude sort of sensation still persists and which therefore cannot be ascribed to the trigeminus. As the chorda tympani is also distributed in this region, it is fair to assume that the mild preservation of tactile

sense is due to the presence of sensory fibers in the chorda. This theory is still further strengthened by one of his cases, in which after an extirpation of the Gasserian ganglion, a facial palsy supervened. Before the onset of the palsy and after the removal of the ganglion, a cotton swab swept over the anesthetic trigeminal area was distinctly felt and gave rise to a crude sort of sensation. This slight persisting sensibility vanished with the appearance of the facial palsy, which would indicate the presence of general sensory fibers in the chorda of facial origin.

Another group of cases has sometimes been cited in support of the theory that the seventh nerve may carry sensory fibers to the face. (J. K. Mitchell (25).) I refer to certain abnormalities in the area of anesthesia after section of one or more branches of the trigeminus. In some instances the resulting area of anesthesia has been so irregular or so transitory as to awaken the suspicion that some other sensory system also participated in this innervation. These cases are uncommon, and are not confirmed by the studies of the field of anesthesia in the total ganglion extirpations. When they do occur, a more satisfactory explanation would be physiological variation and overlap or a restoration of function by regenerative processes.

Spiller (26) also observed after extirpation of the Gasserian ganglion in certain cases that a distinct pressure sense was preserved in the anesthetic area, in which all other forms of sensation were lost. Ivy and Johnson (27) confirmed this observation; and in a case of Gasserian ganglion extirpation found that light touch, pain and temperature sensations were lost in the trigeminal area, but that deep pressure sensations were retained, a manifestation which might lead one to infer that the removal of the ganglion had been only partially successful.

Following the hypothesis of Henry Head that the motor nerves convey sensory fibers to the muscles and tendons (deep sensibility); they ascribe this preservation of the pressure sense to the presence of such fibers in the motor filaments of the facial.

This brief sketch outlines in a general way the various disturbances of a sensory nature which have been described in the course of the clinical development of the facial nerve, *i. e.*, the pain of facial neuritis, the hypesthesias of the face and mucous membrane, the area of anesthesia in the concha and the slight hypesthesia in the chorda tympani distribution of the tongue.

Notwithstanding the occurrence of these sensory symptoms and the various proofs furnished by embryology and anatomy of the mixed character of the seventh cranial nerve, no systematic attempt has been made to attach to this system a general sensory function. On the contrary, these symptoms and others of a similar sensory character, have been assigned to sensory systems of adjacent nerves.

As an introduction to my own views on this subject, I will refer briefly to the anatomy of this region and the recent accessions to our knowledge of the seventh nerve; and having outlined the probable extent and distribution of the sensory facial, I will proceed to a discussion of its general sensory functions and symptoms.

Anatomical Considerations

It may be asserted as a definitely established fact that the facial is a mixed nerve, having the same morphological and developmental significance as the other mixed cranial nerves; the trigeminus, glosso-pharyngeal and the vagus. In common with these nerves, it possesses a ganglion composed of unipolar cells; the central processes of which terminate in the *fasciculus solitarius* of the medulla, in the same manner as do the central processes of the ninth and tenth nerves. These central fibers constitute the sensory root of the seventh nerve and grouped together form the *pars intermedia* of Wrisberg. On the distal side of the ganglion the intermediate fibers are supposed to be continuous with the chorda tympani and as such are associated with the special sense of taste on the anterior two-thirds of the tongue.

Sappolini at one time attempted to establish a separate system composed of the nerve of Wrisberg, the geniculate ganglion and the chorda tympani, and this has sometimes been termed the thirteenth cranial nerve of Sappolini; but the fusion of the chorda fibers with the motor trunk of the facial does not permit of such a morphological separation, and the thirteenth cranial nerve has not been accepted by anatomical writers.

The facial in its course through the Fallopian aqueduct, that is, from the level of the geniculate ganglion to its exit at the stylo-mastoid foramen, gives off numerous collateral branches. The functions of some of these branches, more particularly of the

motor branches and the chorda tympani, are well known; of others the views are conflicting and unsatisfactory.

These branches I will now take up in the order of their origin, with remarks upon their probable sensory functions.

From the geniculate ganglion itself there arise two branches which are called the great and small superficial petrosal nerves; these pass through separate bony foramina and terminate in important ganglia of the cerebral sympathetic system (Meckel's ganglion and the otic ganglion). Both of these petrosal nerves

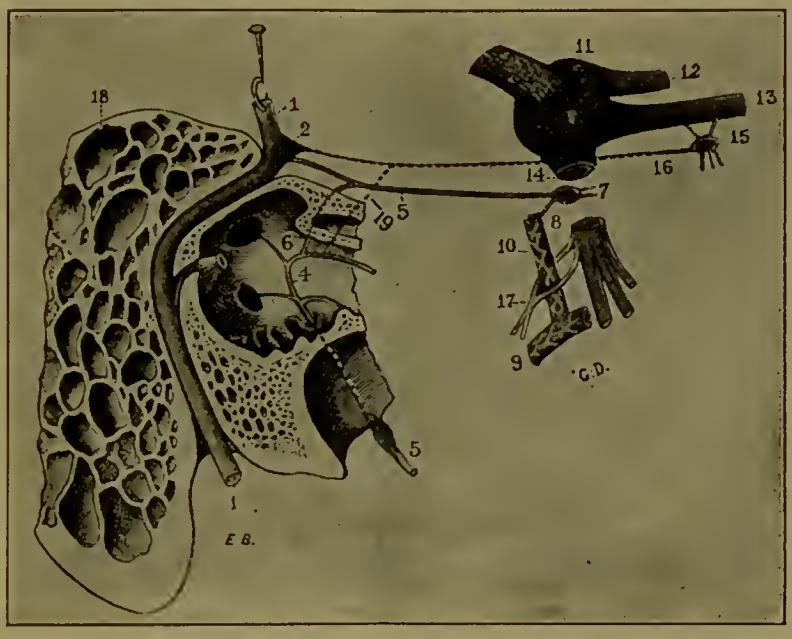

FIG. 2. (Testut's Anatomy.) Showing the facial nerve, geniculate ganglion and the petrosal nerves. 1, Facial nerve; 2, Geniculate ganglion; 3, Glosso pharyngeal, and 4, Jacobson's nerve; 5, Small superficial petrosal nerve; 6, Small deep petrosal nerve; 7, Otic ganglion, and 8, its sympathetic root; 9, Internal maxillary artery; 10, middle meningeal artery; 11, Gasserian ganglion and its three branches; 12, Ophthalmic; 13, Superior maxillary; 14, Inferior maxillary; 15, Spheno-palatine ganglion (Merkel's); 16, Great superficial petrosal or Vidian nerve; 19, Great deep petrosal nerve; 18, Mastoid cells; 17, Auriculo-temporal branch of Fifth.

give off in their course descending branches which take part in the formation of the tympanic plexus; these descending branches are called the great and small deep petrosal nerves. (Fig. 2.)

Many diverse and conflicting functions have been assigned to the petrosal nerves. They are supposed by some writers to transmit the fibers of the chorda tympani from the geniculate to the trigeminus nerve; by others to convey motor impulses from the facial to the spheno-maxillary region, and retrograde sensory

fibers from the trigeminus backwards to the facial as well. Some of these functions have already been disproved and abandoned, and others have no definite proof and are purely theoretical. Cushing (28) in his studies of the trigeminal field of anesthesia after Gasserian ganglion extirpation has shown conclusively that the chorda fibers do not pass to the brain in the trigeminus as was originally held. Furthermore, the supposed motor function of the great superficial petrosal nerve (Vidian nerve) in its relation to the levator palati muscles was disproved by Rethi, who has shown that this function is subserved by the vagus. Furthermore, there is neither clinical nor embryological evidence to show that fibers of common sensation pass from the fifth to the seventh nerve by the petrosal branches.

Dixon (29) has clearly demonstrated the sensory nature of the Vidian or great superficial petrosal nerve and its developmental relation to the cells of the geniculate ganglion. The small superficial petrosal likewise takes its origin from the geniculate ganglion, sends a descending branch to the tympanic plexus and terminates in the otic ganglion of the cerebral sympathetic system; the sensory nature of which is not less evident.

The facial nerve proper which courses within the Fallopian aqueduct, in addition to motor chorda fibers, contains a certain number of sensory fibers as well, which spring from the cells of the geniculate ganglion. The presence of such sensory fibers in the facial trunk having been demonstrated experimentally by Amabolino. These experiments consisted in cutting the nerve at its exit from the stylo-mastoid foramen, and studying the retrograde degenerations in the cells of the geniculate ganglion; another proof that these cells have a common sensory function apart from those subserving the function of taste. These sensory fibers in the trunk are destined for the cutaneous distribution of the facial on the external ear (zoster zone of the geniculate). Furthermore, it has been shown that the chorda tympani in some cases gives off a small branch to the tympanic plexus and we have already learned from the hypesthesia of the tongue in facial palsies that the chorda conveys common sensory fibers to this distribution as well. In this connection it is also worthy of mention that the lingual branch of the facial which innervates the stylo-glossus and palato-glossus muscles at the base of the tongue, also sends mucous filaments to the anterior

pillar of the fauces and the adjacent region. And while it is true that the lingual has an anastomosis with the glosso-pharyngeal nerve, there is no proof that these filaments to the mucous membrane are not part of the sensory facial system, although I have not yet been able to demonstrate clinically anesthesia in this area.

The sensory distribution of the facial to the tongue, fauces (?), and tympanic cavity, with its prolongation into the eustachian tube and mastoid cells, represents the remnants of what in the lower vertebrates is a very considerable sensory distribution to the vault of the palate, tongue, and floor of the mouth

FIG. 3. Showing the distribution of the Cranial nerves in the lower vertebrate forms (Wiedersheim).

(Fig. 3). This at one-time important sensory distribution has in the course of phylogenic development undergone a considerable shrinkage and displacement by the trigeminus. A vestigial remnant in the mouth is still demonstrable, and an important sensory innervation of facial origin still exists in the middle ear and its prolongation and on the external ear.

I have not given in detail the entire array of facts, clinical and anatomical, on which my conclusions are based, but have merely outlined in a general way the results of the more recent investigations. Supported by these facts I would regard the sensory distribution of the facial to be as follows: To the tympanic cavity and its prolongations into the mastoid and eusta-

chian tube by the petrosal nerves; a vestigial innervation to the tongue and tonsillar region through the chorda tympani and lingual (?) branches, and a cutaneous innervation in the interior of the auricle by fibers which pass out with the trunk, some of which probably reach their destination through the posterior auricular branch of the seventh (Valentin (30)).[2] (Fig. 4.)

Our present anatomical nomenclature of this region is so

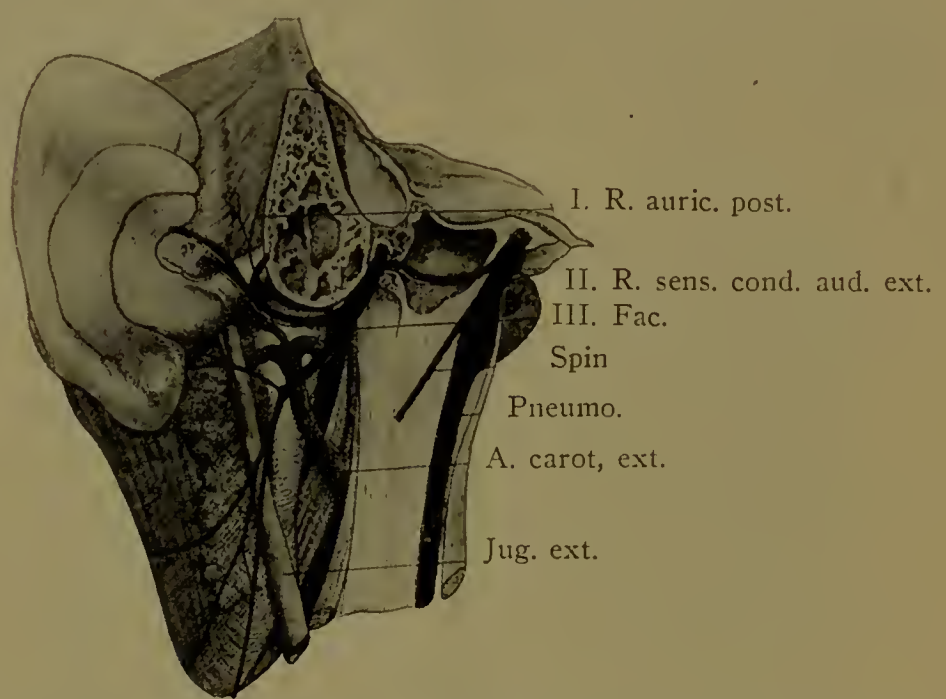

FIG. 4. (Poirier et Charpy.) A posterior view of the facial nerve at its exit from the stylo-mastoid foramen (III), showing the posterior auricular branch of the Seventh (I) and the auricular branch of the vagus (II) as it transverses the Fallopian aqueduct.

very perplexing and confusing that a revision of some sort would seem justified from the clinical standpoint. The one which has suggested itself to me, while perhaps, not satisfactory for anatomical descriptions would, at least, simplify the matter for clinical purposes, and would unite in a practical way the sensory and motor systems of the seventh nerve and its connections with the central sympathetic. With this end in view I would regard the motor and sensory systems of the facial as a single nerve, the seventh cranial nerve, having a ganglion (the geniculate) and two roots (motor and sensory). The motor root is the facial nerve proper to the point of its fusion with the gan-

[2] Valentin has described cutaneous filaments to the auricle, from the ascending and horizontal branches of the posterior auricular branch of the facial.

glion; the sensory root is the so-called *pars intermedia* of Wrisberg. In this respect it is the homologue of the fifth nerve with its Gasserian ganglion, sensory and motor roots. On the distal or peripheral side of the ganglion, at which point there is a fusion of the motor and sensory roots, I would divide the facial system into three principal branches. This would not only simplify the nomenclature of this region, but would give to the sensory facial the importance which it deserves. It is also justified by anatomical distribution, the facial system breaking up into these divisions at the geniculate level, all of which are quite separate in their course and distribution.

The first branch of the seventh nerve on the distal side of the ganglion is the great superficial petrosal which emerges from the tip of the geniculate, passes through the hiatus Fallopii and terminates in Meckel's ganglion which is attached to the second division of the fifth. In its course it gives off the descending branch to the tympanic cavity and enters into the formation of the tympanic plexus. The first branch then connecting the geniculate ganglion with the middle ear, and with the sphenopalatine ganglion of the cranial sympathetic system, constituting its *ramus communicans*. This branch of the facial system stands in relation with the superior maxillary or second division of the fifth, and has important reflex functions (reflex otalgia). (Fig. 2.)

The second branch of the seventh is the small superficial petrosal nerve, takes its origin from the lower portion of the geniculate ganglion, enters a separate bony canal and terminates in the otic ganglion of the cerebral sympathetic system which is attached to the third division of the fifth. Like the great superficial petrosal it gives off in its course a descending branch to the tympanic cavity which enters into the formation of the tympanic plexus.

The second branch then connects the geniculate with the middle ear and with the cerebral sympathetic constituting its *ramus communicans,* and has important reflex functions from the connection with the third division of the fifth (reflex otalgia).[3] (Fig. 2.)

[3] It is possible that both of these petrosal branches may carry motor fibers from the facial to the spheno-maxillary region as well, but this is merely speculative and has not been established.

The third branch of the seventh is the classical motor nerve as it passes through the Fallopian aqueduct. Around this branch is grouped the well established symptomatology of Bell's palsy. This branch contains motor fibers proper, the chorda tympani branch to the tongue and sensory fibers which emerge at the stylo-mastoid foramen for distribution on the auricle. (The zoster zone of the geniculate.)

In such a reconstruction of the facial system into sensory and motor roots, ganglion and three peripheral branches, the first and second divisions bring the geniculate into relation with the

FIG. 5. Anastomoses of the facial and auditory nerves. 1, facial; 2, auditory; 3, nerve of Wrisberg; 3^1, geniculate ganglion; 4, internal anastomoses; 5, external anastomoses. (Testut.)

middle ear and the cerebral sympathetic system and into reflex relationship with the trigeminus, and furnishes an anatomical basis for reflex ear pains, or otalgias, following deep-seated ulcerations in naso-pharynx and buccal cavity.

The third division, in addition to its motor and taste functions, conveys sensory fibers to the tongue, to the auricle, and possibly to the region of the tonsil through the lingual branch. I would also emphasize the presence of certain anastomotic branches which pass between the geniculate ganglion, the nerve of Wrisberg and the auditory nerve in the depths of the internal auditory canal. The function of these anastomoses is unknown, but their sensory character, and their origin in cells of the geniculate ganglion is generally accepted, and it is very probable that

they represent a sensory innervation of the auditory nerve (internal ear?).[4] (Fig. 5.)

I have refrained in these general anatomical considerations from any elaborate enumeration of the facts furnished by embryology and anatomy, which support my views on the sensory system of the facial. There is, however, one series of investigations which I would mention in its relation to the cell function of the geniculate ganglion. As I have previously remarked, the fibers of the chorda tympani originate in these cells and according to very good authority pass in the nerve of Wrisberg to the medulla; so that a part of the function of this ganglion must be concerned with the sense of taste; but that this is not the sole sensory function of this structure has been demonstrated by the histological investigations of W. His, Jr. (32). His counted the fibers in the chorda tympani and the ganglion cells in the geniculate of a 22 mm. human embryo. By actual count it was ascertained that while the chorda contained about one hundred and twenty fibers there were seven to eight hundred ganglion cells; the sensory functions of these other cells are unknown. If one considers as well that cells and groups of cells of exactly the same type are to be found scattered along the entire length of the nerve of Wrisberg, the petrosal nerves and the trunk of the facial itself, it must be admitted that there exists a very considerable sensory system belonging to the seventh nerve, which is not concerned with the sense of taste and which probably subserves the other general sensory functions of this nerve.

That the function of this sensory mechanism should so long have escaped detection is not surprising. We had to do with a small ganglionic system intercalated between the large system of the trigeminus in front and the systems of the glosso-pharyngeal and vagus behind. As a result there were numerous anastomoses of which it was very difficult to determine their motor or sensory nature and their proper ganglionic origin. The subject was still further complicated by the intricate relations of the facial system to the highly specialized structures of the auditory mechanism. Indeed it is only to the study of the simple

[4] Erlitki (31) has suggested that certain cells and groups of cells found in the course of the auditory nerve near its termination send their central processes to the nerve of Wrisberg.

structural types of lower vertebrate forms that we owe our present knowledge of the mixed nature of the seventh nerve and its relation to the first branchial or ear cleft.

That its distribution on the auricle was not recognized is also easily understood. The area is small and because of its situation in the auricular folds and the canal is difficult of access. Furthermore, the concentration of the auricular branches of the fifth, ninth, tenth and upper cervical nerves in the same region still further complicated matters, so that a small area of anesthesia in this region would rapidly fade in the overlapping of adjacent marginal areas. (As we have already seen Gowers demonstrated such an anesthesia in early and severe cases of facial paralysis, but referred it to the trigeminus.)

With these obstacles to overcome the importance of the herpes zoster method as a means of determining its ganglionic representation requires no emphasis. The importance of this method has already been demonstrated in the spinal ganglia (Head and Campbell (33)), and how much greater would be its significance in the case of the geniculate; a small ganglion of unknown representation in which the cutaneous distribution was small, difficult of access and of demarcation by the usual anesthetic methods.

Sensory Symptoms and Syndromes of the Seventh Nerve

(*a*) *Herpetic Inflammations of the Geniculate Ganglion. (Herpes Oticus.)*—In the historical summary I have already enumerated various sensory symptoms in facial palsy, which have accumulated during the past few years. In the anatomical résumé I have shown the existence of a sensory mechanism in the seventh nerve entirely apart from that concerned with the special sense of taste, and have indicated its probable range of distribution, *i. e.,* the auditory nerve and its terminations, in the internal ear; the tympanic cavity and its prolongations into the mastoid cells and eustachian tube; the interior of the auricle and vestigial remnant on the tongue.

I shall now proceed to an elaboration of the sensory symptomatology, and will begin with the herpetic inflammations of the geniculate ganglion. As I have already treated this subject at length in other communications (34), I shall simply outline the symptomatology in order to give completeness to my remarks.

The geniculate, like other ganglia of the spinal type, originating as outgrowths of the neural ridge, may be the seat of the specific inflammation of herpes zoster, the *herpes oticus.* Like the other sensory symptoms of facial origin, the herpes oticus had been referred to the neighboring systems of the trigeminus and the upper cervical nerves.

In herpetic inflammation of the geniculate, with the general symptoms of zona there is pain, localized in the depths of the ear, auditory canal and mastoid region (herpetic otalgia).

Following the pain there appears the characteristic eruption of zoster which is distributed in the canal or on the external surface of the auricle.

The zoster zone for the geniculate is not as yet definitely outlined, but it lies within a cone-shaped area, represented by the tympanic membrane, the walls of the auditory canal, external meatus, concha, tragus, antitragus and antihelix.[5]

In one of my cases belonging to this group in which histological studies were made, well marked degenerative changes were found in the intra and extra medullary portions of the sensory root of the seventh, the nerve of Wrisberg.

Herpes oticus is not, however, the only manifestation of geniculate inflammation, but from the close proximity of the facial and the auditory nerves, these structures may be involved, which lends a special interest to this localization of the disease. So that with the herpes oticus there may be facial paralysis and symptoms referable to the acoustic nerve. In severe cases the auditory complication may give rise to the typical symptoms of Ménière's disease; vertigo, vomiting, nystagmus, disturbances of equilibrium, tinnitus aurium and deafness (herpes oticus with facial paralysis and auditory symptoms).

Another group of cases must also be mentioned in which the same neural complications (seventh and eighth nerves) occur with herpes zoster of the face and neck (herpes zoster facialis and occipito collaris).

This group of cases may be explained by the tendency of the

[5] The vagus and gloss-pharyngeal ganglia both have a representation within this area as well. I have seen two cases in which the eruption was distributed posteriorly in the cleft between the auricle and the mastoid process, in the distribution of the posterior auricular branch, so that there is probably considerable variation in the ganglionic representations (zoster zones) of the seventh, ninth and tenth nerves in the region of the auricle.

specific toxin of zona to produce inflammatory reactions in a series of ganglia, although the chief or *eruptive focus* is confined to a single ganglion.

In this manner the geniculate may be the seat of an inflammatory reaction when the eruptive focus is in the Gasserian or upper cervical ganglion, which may extend to the adjacent seventh and eighth nerves (herpes facialis and herpes occipito collaris with facial palsy and auditory symptoms).

That the geniculate ganglion may be encroached upon from without, and secondarily involved, inducing pains and an eruption of herpes on the auricle is rendered very probable by a case of endothelioma of the petrous bone which came under my observation.[6]

(*b*) *Neuralgia of the Seventh Nerve* (*Otalgia* (36)).—In discussing the distribution of the pains following herpetic inflammations of the geniculate, we have seen that they are very definitely localized in the structure of the auditory mechanism (the herpetic otalgia).

In addition to the pain of organic origin there must also be considered a group of pure sensory neuroses of the seventh cranial nerve, under which heading I include certain definite clinical types of otalgia. By otalgia I refer to a large group of cases in which pain is definitely localized in the auditory mechanism, *i. e.*, the depths of the ear, the mastoid region, the canal and on the surface of the auricle. As might be surmised from the highly specialized structure of the auditory mechanism and its exposed situation through the medium of the auditory canal and the eustachian tube, such neuralgic affections are not uncommon. Schwartze gives the proportion of non-inflammatory ear-ache to that of inflammatory origin as 1.8 per cent. Considerable confusion prevails in the grouping of these cases and no very definite or satisfactory anatomical basis has as yet been furnished. The otalgias have been indiscriminately classed as neuralgia of the tympanic plexus, the plexus of the eustachian tube; or if the

[6] It is also very probable that the ganglion of the auditory nerve may be primarily involved in zona. The cells of the ganglion spirale and the ganglia of Scarpi and Boettger take their origin from the neural ridge, from which are developed the ganglion of the spinal type; and although they are morphologically different by reason of retaining their primitive bipolar character, they are genetically the same, and as such probably liable to herpetic inflammations. There is also clinical evidence to support this theory (35).

pain was situated in the auricle it was relegated either to the trigeminus in front or the cervical nerves behind.

While it is true that ear pains may occur as a part of the trifacial or occipito-cervical neuralgia (auricular branches), one can hardly conceive of a pain sharply localized in the auditory mechanism as the sole manifestation of a tic douloureux of the fifth, or of the occipito-cervical nerves. The same is true of the glosso-pharyngeal nerve, which while it participates in the innervation of the tympanic cavity (Jacobson's nerve), has an extensive innervation in the region of the tonsil, the palate and upper pharynx. I would therefore particularly emphasize the existence of a well defined clinical type of neuralgia, dependent upon a neurosis of the geniculate ganglion and its system, and which deserves to be ranked with the other time-honored and classical groups of this affection (38).

The sensory system of the facial alone has a circumscribed distribution in the auditory mechanism. That pure otalgia is a sensory manifestation of the geniculate system is still further corroborated by the distribution of the herpetic pains in cases of inflammation of the geniculate ganglion. These pains are very definitely otalgic in their distribution, and in severe cases before the appearance of the eruption, have simulated so closely those of otitis media, that the tympanum has been incised for the relief of this condition. The pains of otalgia are typically neuralgic in character. A very classical description of a case of "tic douloureux of the ear" is contained in Nottingham's (37) text-book published in 1857, and which he referred to the trigeminal system.

Furthermore, I believe that cases of this type are much more frequent than might be inferred from a perusal of the literature, and other therapeutic measures failing the advisability of some form of surgical intervention would deserve consideration, similar to the methods employed in the treatment of tic douloureux of the face. An intermittent type has also been described with the curious periodic character sometimes observed in supraorbital neuralgia and which responds to quinine.

In addition to the idiopathic and herpetic otalgias, a *reflex* form must also be recognized. In it there occurs a sharply localized pain in the ear as a result of deep-seated ulcerative affections in the buccal cavity and naso-pharynx. Caries of the

teeth is an especially frequent cause. Otalgia occurring as a result of irritative lesions in this region is to be regarded as a referred pain and having the same significance as the visceral referred pains. The naso-pharynx and buccal cavities receive fibers from the large sympathetic ganglia on the second and third divisions of the fifth (Meckel's ganglion and the otic ganglion). Both of these are in direct communication with the geniculate through the superficial petrosal nerves, which may be regarded in the nature of *rami communicantes,* similar to the connections existing between the spinal ganglia and the splanchnic system.

Tabetic Otalgia.—In addition to the clinical types already mentioned, I believe that we must also recognize as occurring in the course of tabes dorsalis, lancinations in the auditory mechanism dependent upon degenerations in the nerve of Wrisberg (tabetic otalgia). As might be supposed these cases occur more frequently in the cephalic or bulbar types of this disease. In this localization of tabes, degenerations have already been demonstrated in the sensory roots of the fifth, ninth and tenth nerves, but so far as I am aware the nerve of Wrisberg has escaped attention, although it is a sensory root, springing from the cells of the geniculate ganglion.

I have examined a large number of tabetics in order to determine the presence of ear pains which might be referred to the sensory system of the seventh nerve. Up to the present time I have found five cases in which there were sharp lancinations like the prick of a needle in the depths of the ear. In all these, the lancinations were very sharp and were frequently accompanied by a sudden reflex jerk of the head. In all, organic disease of the ear was excluded. In one of these cases a very advanced tabes of fifteen years duration, I found well marked degenerations in the sensory root of the seventh (nerve of Wrisberg), which shows conclusively that this structure may be involved in the root degenerations of tabes. I would also direct attention to the occurrence of a kind of aural or tympanic *crisis,* and its probable relation to the sensory system of the seventh. These attacks are characterized by sharp lancinating pains in the ear, followed by tinnitus, vertigo and transient disturbance of hearing.

(*c*) Sensory Disturbances in Facial Neuritis.

As was pointed out in my preliminary remarks, various sensory disturbances have been noted in cases of facial neuritis. I will now discuss this aspect of my subject on the basis of thirty personal cases. By facial neuritis I refer to the common form of Bell's palsy, in which the nerve has been involved in the Fallopian canal between the geniculate ganglion and the stylo-mastoid foramen. This division of the nerve as we have seen is composed of motor fibers, the chorda tympani and sensory filaments destined for the external ear. (The chorda also sends a filament to the tympanic plexus and general sensory fibers to the tongue.)

If this section of the facial becomes the seat of an inflammatory process, the pressure effects would be very much augmented by its confinement within the bony canal and there would, if the sensory fibers became involved, result necessarily pain.

This varies greatly in character, duration, and localization in the different cases. In my series this symptom was present in all but two. Usually it was an early symptom and preceded the onset of the palsy, but in a few instances followed in the wake of the paralysis. As has been indicated by previous observers, it is chiefly centered in the canal and depths of the ear and the mastoid region. It is occasionally referred beneath the lobule, and in some of my cases was localized on parts of the external surface of the auricle, such as the tragus and the margin of the helix. In the more severe forms it radiates to the face, occiput and temporal region. The presence and character of this pain is so well known that it requires no special comment, except its almost constant presence in my cases, and its etiological relation to the sensory fibers in the facial.

It will be recalled that in the Donath series of one hundred and seventy-five cases, pain was mentioned in only seventy-five, but as the greater proportion of Bell's palsy comes under observation after the subsidence of the acute irritative symptoms, this may escape notice unless very specially investigated.

As has been previously mentioned, the hypesthesia of the face was present in only two of my cases, and could only be demonstrated by a most careful comparison with the sound side, and from its nature and distribution can not be regarded as of seventh-nerve origin, or even as anesthesia in the ordinary acceptation of this term. It is rather a functional or physiological

derangement of the fifth-nerve ganglion, induced by the motor insufficiency of the paralyzed face.

In nine of my cases *hypesthesia in the concha* of the ear was demonstrable. In one case the sensibility to touch and pain was increased in this region, and in two cases the aural reflex induced by touching the walls of the external meatus was absent although active on the non-paralyzed side. This disturbance of the sensation in the conchal region never reached the degree of anesthesia; the tactile sense was merely diminished or obtunded in this area, and care was required to demonstrate its presence, but I satisfied myself of its existence in each case by repeated and careful examinations. I was unable to demonstrate any definite changes of sensibility within the canal.

Some years ago Testaz attempted to utilize pain as a symptom of prognostic value in cases of palsy, but subsequent observations have not confirmed its importance. Future investigation may show that the presence of conchal hypesthesia is of value in determining the severity of the lesion to the nerve; for on theoretical grounds the sensory fibers being more resistant would escape in the milder grades of palsy. It is very probable, however, that other factors would have to be considered, which would lessen the importance of this symptom, such as variation and overlap in the adjacent sensory areas of this region. When one considers that the auricular branches of the fifth, tenth and ninth nerves, as well as the upper cervical nerves, converge in the region of the auricle, it is not surprising that the small conchal strip should be lost in compensatory overlapping.

Pain in Primary Affections of the Auditory Nerves.—Before leaving the subject of neuritic pain and its relation to the sensory facial, I would emphasize the connection existing between the nerve of Wrisberg, the geniculate ganglion and the termination of the acoustic nerve—and that pain occurring in the course of primary inflammation and sclerosis of the auditory nerve may be referred to this system. The original *anlage* of the geniculate ganglion and the ganglia of the cochlear and vestibular nerves are united in the ganglion acousticum-facialis, and in the later developmental and structural changes the close relation of these two sensory systems may be traced.

So close is this relationship that some have even regarded the acoustic nerve as a highly specialized part of the afferent facial

system, so that pain in primary affections of the auditory nerve may very justly be ascribed to sensory systems of the seventh nerve.

(*d*) *The Sensory System of the Seventh Nerve as a Reflex Factor in the Causation of Peripheral Facial Twitchings, Myokymia and Spasms.*—Having considered the sensory facial in its relations to herpes zoster, neuritis and neuralgia, it remains to indicate its importance as a reflex mechanism in the transmission of afferent impulses to the facial nucleus.

As we have seen the facial sensory system covers a very considerable area, the central branches of the cells of the geniculate ganglion traversing the internal auditory canal and the base of the skull to the medulla, while the peripheral branches in the petrosal nerves pass to the tympanic cavity and traverse the base of the skull in the middle fossa to the sympathetic ganglia on the second and third divisions of the fifth. Peripheral sensory filaments also course in the petrous canal with the trunk of the facial; so that irritative lesions in the external ear, the middle ear, the internal auditory canal or at the base of the skull would send impulses direct to the nucleus of the seventh by its sensory path. The recognition of this sensory pathway to the nucleus of the facial is, I believe, of practical importance in its etiological relations to the facial spasms of peripheral origin; and should always receive consideration in searching for the primary focus of irritation.

Brissaud, some years ago, separated from the facial twitchings of psychical (tic convulsif) and cortical origin (focal epilepsy), a type which was purely nuclear or peripheral in nature. This he regarded as induced by an irritation of the facial nucleus either directly or indirectly through one of the afferent systems terminating in the medulla near the nucleus and more particularly that of the trigeminus nerve. Irritative lesions originating in the eyes, mouth, nose and teeth, sinuses and other structures innervated by the trigeminus were regarded as important reflex factors. The sensory system of the seventh nerve was not, however, considered as a possible factor.

I have collected a large number of cases of facial spasm of the peripheral type, in which I believe the irritative afferent factor is represented by the sensory system of the facial. Among these may be mentioned structural lesions in the entire auditory

mechanism, *i. e.*, internal, middle, and external ear, also small tumors, aneurisms and localized inflammatory products situated at the base of the skull in proximity to the facial nerve.

It is a well known fact that facial twitchings and contractions occur very frequently as a sequence of the severer grades of Fallopian neuritis. It has also been rarely observed preceding the onset of the paralysis. In all of these groups the irritation may be conveyed directly to the nucleus by the sensory facial. I have also seen typical myokymia of the face, associated with deafness of many years duration, due to extensive lesions of the middle ear.

Babinski who has recently contributed to the symptomatology and pathogenesis of facial spasms believes that because of certain peculiarities in the nature and distribution of the spasm, it must follow irritative or dynamic lesions of the nucleus or trunk of the nerve; meaning trunk in its motor or efferent sense.

It is not necessary to enter into a discussion of the probability or improbability of this hypothesis; that irritation of the motor trunk of a nerve may have as a result, periodical or chronic contractions of a nuclear character. For as we have seen, the motor trunk of the nerve is accompanied by a corresponding sensory system from the medulla to the stylo-mastoid foramen, so that any impulses originating from an irritative or dynamic lesion would be conducted reflexly to the nucleus by its afferent path, and would explain the resulting state of nuclear irritability.

There are still other reflex phenomena of clinical interest, which might be mentioned in connection with the facial system, the trigeminous and the organs of hearing, but this phase of the subject is still vague and uncertain, and I mention it only as a field for future investigation. No satisfactory explanation has as yet been offered for the occurrence of temporary deafness or hyperacousis following disease or extraction of the teeth.

I have already considered the reflex otalgia as a visceral referred pain to the geniculate system; and when one also takes into consideration the close relationship existing between the auditory system and the geniculate system, originating as they do from a common nucleus in embryonal life (ganglion acousticum-facialis), we may find a solution for some of these curious auditory phenomena.

Concluding Remarks

In the foregoing pages I have given a general survey of the clinical development of the facial nerve from a sensory point of view. That this nerve has a sensory system of importance finds corroboration on many sides. The results of investigations in the realms of embryology and anatomy find their counterpart in symptomatology and pathology.

I believe, therefore, that the seventh nerve should take a definite place as a mixed nerve and should rank with the fifth in our symptomatology and nomenclature.

In the case of the trigeminus the sensory functions far outweigh in importance its motor functions, which is in accord with the anatomical relations and distribution of this nerve.

In the case of the seventh nerve, the sensory system while playing a less important rôle, must still be regarded as a very definite factor and furnishes the basis for a variety of symptoms and syndromes of a sensory nature.

The anatomy of this region is so complex and so burdened with long descriptive names that a simpler division of the subject would seem justified, a division such as has been adopted in the case of the fifth nerve. In anatomical descriptions of the fifth nerve there are recognized a sensory and motor root on the proximal side of the ganglion, and three branches or divisions on the distal side.

The *ophthalmic* or first, *superior maxillary* or second, and the *inferior maxillary* or third, the motor root of the fifth joining the inferior maxillary division and is described with it.

This anatomical division of the trigeminal system has proved to be a very practical one and it has seemed to me that a reconstruction of the facial system along similar would have a clinical value (Fig. 6).

Such a reconstruction would recognize on the proximal side of the ganglion a motor and a sensory root and on the distal side of the ganglion three branches:

Peripheral Divisions of the Seventh Nerve.

(1) The great superficial petrosal with its tympanic branch and connections with Meckel's ganglion.

(2) The small superficial petrosal nerve with its tympanic branch and connections with the otic ganglion.

(3) The Fallopian facial, including the motor trunk, the chorda tympani and sensory fibers for the auricle.

While such a division of the nerve may not serve the anatomist, who divides the facial according to its relations in the

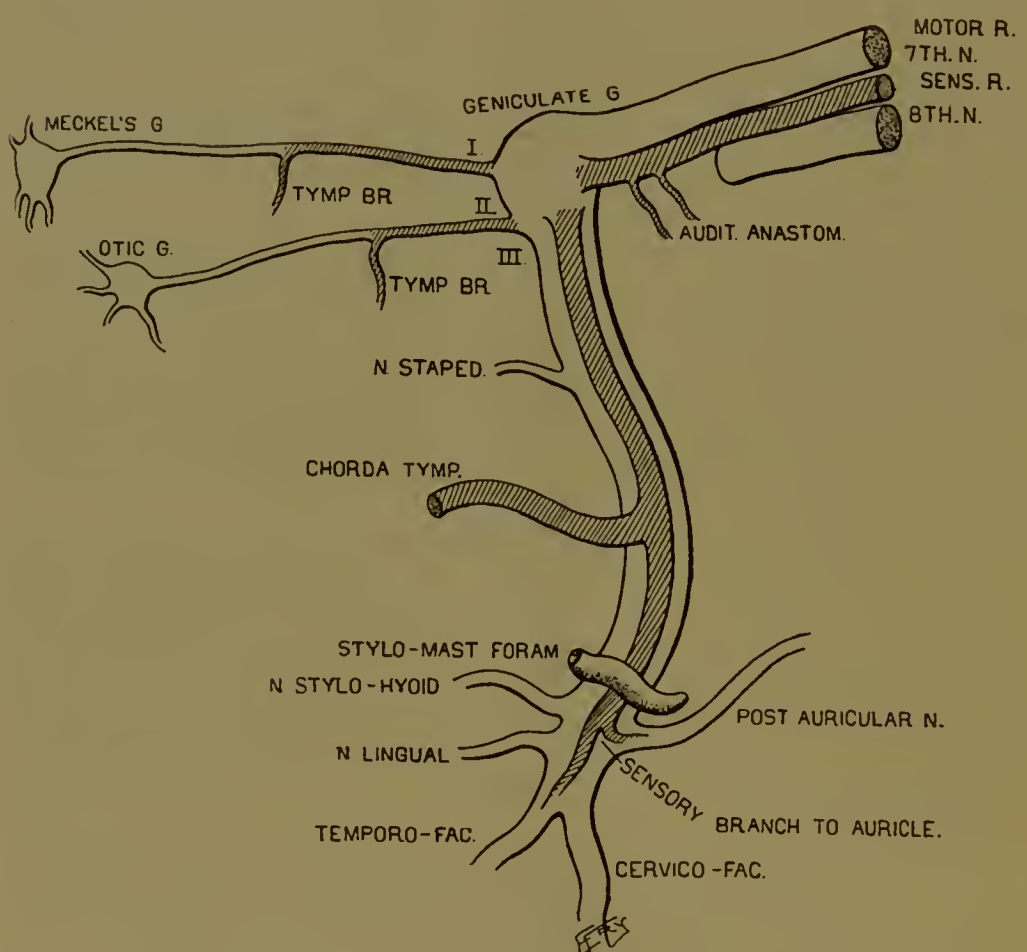

FIG. 6.

temporal bone into intra- and extra-petrous branches, it would, however, furnish a rational anatomical basis for clinical descriptions and the classification of the various motor, sensory, special sense and splanchnic functions of the seventh cranial nerve. (Figs. 5 and 6.)

SUMMARY.

Sensory Functions of the Seventh Nerve.

Special sense fibers to the anterior two thirds of the tongue (chorda tympani).

Sensory anastomosis with the terminations of the auditory nerve (internal ear).

Sensory fibers to the middle ear, mastoid cells and eustachian tube (deep branches of the petrosal nerves).

Sensory fibers to the anterior two thirds of the tongue (chorda tympani).

Sensory fibers to the external ear (emerging with the facial trunk at the stylo-mastoid foramen).

Sensory Symptoms and Syndromes of the Seventh Nerve.

PAIN.—*Organic Origin.* Fallopion neuritis (third branch); tabetic degenerations (sensory root); herpetic otalgia (geniculate ganglion).

Functional Origin.—Primary otalgia (tic douloureux of the ear); reflex otalgias.

Anesthesias.—Hypesthesia of the concha; hypesthesia of the anterior two thirds of the tongue; ageusia of the anterior two thirds of the tongue.

Reflex.—Reflex etiological factor in the production of reflex facial twitchings and spasms.

Syndromes.—The herpetic inflammations of the geniculate ganglion; herpes oticus; herpes oticus with facial palsy and acoustic symptoms; herpes facialis and herpes occipito-collaris with seventh palsy and acoustic symptoms.

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